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Intralobar pulmonary sequestration in an adult female patient mimicking asthma: A case report

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ABSTRACT

Pulmonary sequestration (PS) is a rare congenital broncho-pulmonary malformation. The main feature of this disease is that partial lung tissues separate from the main lung during the embryonic period, receiving blood supply from systemic circulation arteries. Pathogenesis of PS is not clear, and categorized into congenital and acquired PS. We report a case of a 38 year old woman smoker with medical history characterized by difficult to treat asthma with frequent exacerbations and infections since childhood. CT scan showed a partial PS of left lower lobe, supplied by an abnormal artery arising from supra-diaphragmatic aortic diverticulum. Surgical treatment through a lung sequestrectomy and laterobasal segment resection was performed.

1. Introduction

Firstly described in 1861 by Rokitanski and Rektorzik, Pulmonary Sequestration (PS) encompasses a spectrum of congenital lung anomalies exhibiting anomalous systemic arterial supply to the lungs and/or abnormal connection between one or more of the major components of lung tissue [1]. In 1946, Pryce introduced the term 'sequestration' to describe congenital abnormalities characterized by an anomalous systemic arterial supply to the lung and atresia or hypoplasia of the pulmonary artery [2]. In detail sequestration was defined by Pryce as a "disconnected (dislocated, ectopic) bronchus–pulmonary mass or cyst with an anomalous systemic blood supply" [2]. The incidence of PS is 0.29% accounting for 0.15–6.4% of all congenital pulmonary malformation. PS mainly arises from an abnormal budding of primitive foregut as well as other organs congenital malformations [3–6]. In agreement with Pryce's nomenclature, PS is an area of lung parenchyma not connected with the bronchial system and supplied by an abnormal artery arising from the aorta or one of its side branches. The arterial supply is derived in 75% of cases from thoracic aorta, in 20% from abdominal aorta and in 15% from two different origins. The venous drainage is mainly via pulmonary veins [7,8]. PS is classified as intra-lobar pulmonary sequestration (IPS) or extra-lobar pulmonary sequestration (EPS) [9,10]. IPS is incorporated within normal lung tissue and the venous drainage is usually to the pulmonary circulation [9]. EPS is separated from normal pulmonary tissue by its own visceral pleura and the lesion is located outside the lung; in EPS the venous drainage is usually to the systemic circulation [10]. While in IPS the posterior basal segment is most often affected, 90% of EPS affects the left lower lobe. PS clinical manifestations involving respiratory and cardiovascular system (e.g. fever, cough, sputum occasionally purulent, chest tightness, wheezing, regurgitation, cyanosis, hemoptysis) are not specific. Therefore, misdiagnosis with other pathologies such as tuberculosis, pneumonia, pulmonary cyst, lung abscess, pulmonary tumor, pectus excavatum as well as asthma is reported [11]. Asthma is a heterogeneous inflammatory disease characterized by airflow limitation and bronchial hyper-responsiveness which originates from complex interactions between individual and environmental factors [12–22]. Gold standard method for PS diagnosis is angiography but optimal identification of parenchymal and vascular structures

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involved in sequestration is obtained by CT scan [23,24]. Adult pulmonary sequestrations are usually treated with minimally invasive techniques; to control symptoms and avoid hospitalizations early surgical management is recommended [25]. Nevertheless, adult stem cell-based therapy is a promising novel approach for treatment of lung injury. Since the finding of putative human endothelial progenitor cells, human CD34 progenitor cells isolated from bone marrow, peripheral blood and cord blood have been tested in many preclinical models of vascular and tumoral diseases [26–31].

2. Case report

We report a case of a 38 year old woman smoker (20 pack years). The patient was referred to our chest department from a surgical unit for an incidental detection of lung consolidation during preoperative assessment for a breast fibroadenoma. Medical history revealed the presence of difficult to treat asthma with frequent exacerbations and infections since childhood in addition to arterial hypertension. A chest radiograph showed an abnormal pulmonary density of the left lung. The patient complained of a one month history of pyrexia unresponsive to antibiotics. Chest examination revealed widespread expiratory wheeze and inspiratory rales in the base of left lung. The biochemical exams showed raised inflammatory marker (erythrocyte sedimentation rate 46 mm/1 h); arterial blood gas analyses showed a mild hypoxemia (pO^2 76 mmHg, pCO^2 34 mmHg, pH 7,44, HCO^3-25); Spirometric exam confirmed lung obstruction. Acute inflammation with purulent lung secretion from lower left lobe was proved by fibrobronchoscopy, with preserved bronchial canalization. No pathogens were detected by microbiological analysis of sputum and bronchial aspirate. CT scan showed reactive lymphadenopathy with a partial PS of left lower lobe, with excavation areas and lung colliquation (Fig. 1). PS was supplied by an abnormal artery arising from a supradiaphragmatic aortic diverticulum (Fig. 2). We performed a lung sequestrectomy through an intrapleuric thoracotomy at VI intercostal space. We isolated lung tissue and abnormal aortic diverticulum with double vycril 3/0 and overlock with prolene 3/0 running. Finally we resected laterobasal segment and a paraortic lymphadenectomy of 11th station was made.

3. Discussion

Intralobar pulmonary sequestration is located within the visceral pleura and it is more frequent in the left lower lobe (2/3 of cases). It is the most common form of sequestration and shows a higher frequency in males than females (6:1) [1–4]. Bilateral pulmonary sequestration and association of intra and extra-lobar forms of sequestration are very rare to find in literature as are associated malformations suchb as bronchogenic cyst. Symptoms typically occur in the first 20 years of life, characterized by cough, fever and rarely chest pain and hemoptysis [9–11]. Angiography is the gold standard procedure for diagnosis although diagnosis is usually reached, with high diagnostic confidence rate, by volumetric and contrast-enhanced CT scan [23]. CT scan allows identification of parenchymal and vascular structures involved in sequestration such as in our case report [24,32]. Surgery, with thoracotomy or VATS approach, represents the standard care for these patients [25]. To prevent potential serious complications surgical treatment is usually indicated. Surgical procedure consists of removing the injured area preserving the healthy part of the lobe. Technical difficulties such as vessel isolation or risks due to the misunderstanding of the pathological area might justify the choice of one of the two surgical approach? or the decision to

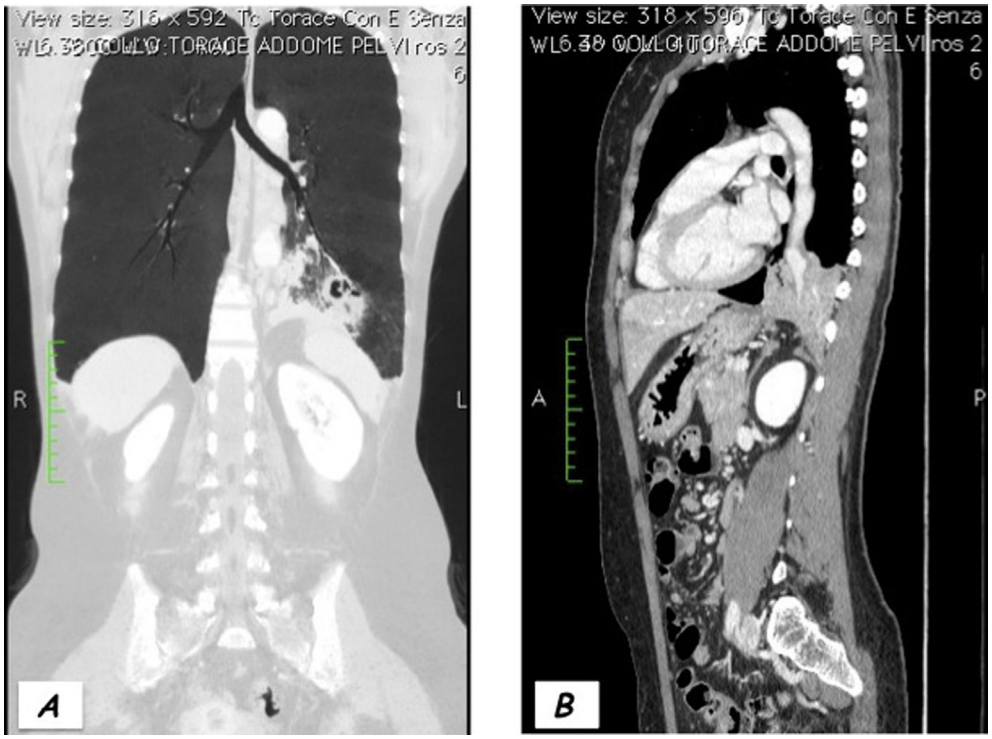

Fig. 1. Contrast-enhanced lung CT scan report partial Pulmonary Sequestration of left lower lobe, with excavation areas and lung colliquation. The feeding artery of sequestrations, arise from a supradiaphragmatic aortic diverticulum – Coronal (A) and Longitudinal (B) view.

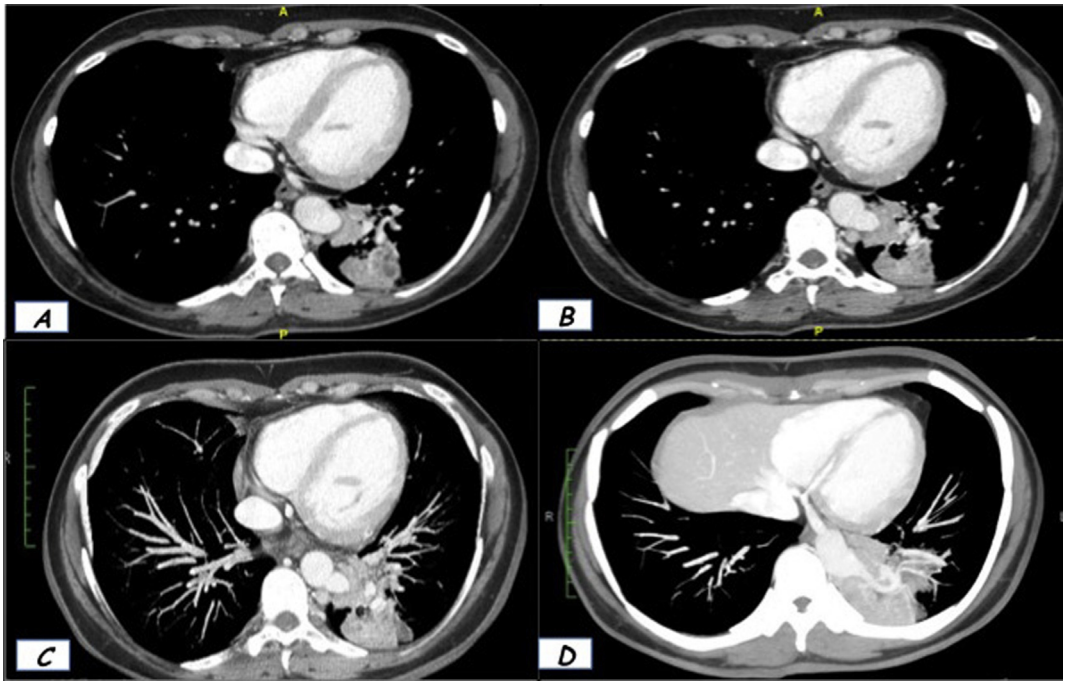

Fig. 2. Contrast-enhanced lung CT scan report partial Pulmonary Sequestration which arterial supply arise from a supradiaphragmatic aortic diverticulum – axial view (A–B) and Maximum Intensity Projection (MIP) (C–D) view.

remove the entire lobe [33,34]. Endovascular embolization of the arteries involved in sequestration might be an optional treatment alternative to surgery such as described by several authors [35].

4. Conclusions

Pulmonary sequestration is a rare and difficult to diagnose lung disease [36]. Common symptoms of PS include recurrent episodes of fever, chest pain and cough. Due to not specific clinical presentation, PS recognition may be delayed and differential diagnosis may result challenging [36,37]. Our observation arise the concept that also difficult to threat asthma should be considered in differential diagnosis. In light of clinical features including frequent infectious exacerbations, asthma cannot be excluded as cause of misdiagnosis of PS [38–42]. Crucial for diagnosis is the contrast-enhanced lung CT scan which reproduces a detailed assessment of the vascular tree structure allowing exclusion of other diseases like neoplasms and smoking related diseases [43,44]; best surgical strategy for PS should guided by CT scan findings [23–25]. When PS is associated to lung cancer [37], Ca^{2+} toolkit changes, that has been observed in other malignancies, including renal cellular carcinoma [45–47], and prostate cancer [48], mielofibrosis [49], could be used as target for selective molecular therapies. We believe that pulmonary sequestration should be considered, even in adults, as an alternative diagnosis to chronic pulmonary disease including asthma.

Conflicts of interest

All Authors have no conflict of interests.

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Ethical approval

Ethical approval was requested and obtained from the “Second University of Naples/AORN Monaldi” ethical committee.

Author contribution

Gennaro Mazzarella: Partecipated substantially in conception, design, and execution of the study and in the analysis and interpretation of data; also partecipated substantially in the drafting and editing of the manuscript.

Carlo Iadevaia: Partecipated substantially in conception, design, and execution of the study and in the analysis and interpretation of data.

Germano Guerra: Partecipated substantially in conception, design, and execution of the study and in the analysis and interpretation of data; also partecipated substantially in the drafting and editing of the manuscript.

Aldo Rocca: Partecipated substantially in conception, design, and execution of the study and in the analysis and interpretation of data.

Nadia Corcione: Partecipated substantially in conception, design, and execution of the study and in the analysis and interpretation of data.

Giovanni Rossi: Partecipated substantially in conception, design, and execution of the study and in the analysis and interpretation of data.

Dario Amore: Partecipated substantially in conception, design, and execution of the study and in the analysis and interpretation of data.

Luca Brunese: Partecipated substantially in conception, design, and execution of the study and in the analysis and interpretation of data.

Andrea Bianco: Partecipated substantially in conception, design, and execution of the study and in the analysis and

interpretation of data; also partecipated substantially in the drafting and editing of the manuscript.

References

[1] B.S. Clements, J.O. Warner, Pulmonary sequestration and related congenital bronchopulmonary-vascular malformations: nomenclature and classification based on anatomical and embryological considerations, Thorax 42 (1987) 401–408.
[2] D.M. Pryce, Lower accessory pulmonary artery with intralobar sequestration of lung: report of seven cases, J. Pathol. Bacteriol. 58 (1946) 457–467.
[3] H.J. Corbett, G.M. Humphrey, Pulmonary sequestration, Paediatr. Respir. Rev. 5 (1) (2004 Mar) 59–68.
[4] D. Belchis, M. Cowan, K. Mortman, B. Rezvani, Adenocarcinoma arising in an extralobar sequestration: a case report and review of the literature, Lung Cancer 84 (1) (2014 Apr) 92–95.
[5] A. Soscia, G. Guerra, M.P. Cinelli, D. Testa, V. Galli, V. Macchi, R. De Caro, Parapharyngeal ectopic thyroid: the possible persistence of the lateral thyroid anlage, Surg. Radiol. Anat. 26 (4) (2004) 338–343.
[6] G. Guerra, M. Cinelli, M. Mesolella, D. Tafuri, A. Rocca, B. Amato, S. Rengo, D. Testa, Morphological, diagnostic and surgical features of ectopic thyroid gland: a review of literature, Int. J. Surg. 12S1 (2014) S3–S11.
[7] B. Savic, F.J. Bertel, W. Tholen, et al., Lung sequestration: report of seven cases and review of 540 published cases, Thorax 34 (1979) 96–101.
[8] E.S. Erden, T.D. Yetim, A. Balci, A.B. Akcay, S. Hakverdi, M. Demirkose, Intralobar pulmonary sequestration with arterial Supply from two different origins: a case report, Ann. Thorac. Cardiovasc. Surg. 18 (2012) 560–563.
[9] C. Hertzenberg, E. Daon, J. Kramer, Intralobar pulmonary sequestration in adults: three case reports, J. Thorac. Dis. 4 (5) (2012) 516–519.
[10] Y. Wei, F. Li, Pulmonary sequestration: a retrospective analysis of 2625 cases in China, Eur. J. Cardiothorac. Surg. 40 (1) (2011 Jul) e39–42.
[11] J. Ou, X. Lei, Z. Fu, Y. Huang, E. Liu, Z. Luo, D. Peng, Pulmonary sequestration in children: a clinical analysis of 48 cases, Int. J. Clin. Exp. Med. 7 (5) (2014) 1355–1365.
[12] G. Mazzarella, A. Bianco, E. Catena, R. De Palma, G.F. Abbate, Th1/Th2 lymphocyte polarization in asthma, Allergy 55 (Suppl. 61) (2000) 6–9 (Review).
[13] A.A. Fryer, M.A. Spiteri, A. Bianco, M. Hepple, P.W. Jones, R.C. Strange, R. Makki, G. Tavernier, F.I. Smilie, A. Custovic, A.A. Woodcock, W.E. Ollier, A.H. Hajeer, The -403 G–>A promoter polymorphism in the RANTES gene is associated with atopy and asthma, Genes. Immun. 1 (8) (2000 Dec) 509–514.
[14] M.A. Spiteri, A. Bianco, R.C. Strange, A.A. Fryer, Polymorphisms at the glutathione S-transferase, GSTP1 locus: a novel mechanism for susceptibility and development of atopic airway inflammation, Allergy 55 (Suppl. 61) (2000) 15–20.
[15] A.A. Fryer, A. Bianco, M. Hepple, P.W. Jones, R.C. Strange, M.A. Spiteri, Polymorphism at the glutathione S-transferase GSTP1 locus. A new marker for bronchial hyperresponsiveness and asthma, Am. J. Respir. Crit. Care Med. 161 (5) (2000 May) 1437–1442.
[16] V. Esposito, A. Lucariello, L. Savarese, M.P. Cinelli, F. Ferraraccio, A. Bianco, A. De Luca, G. Mazzarella, Morphology changes in human lung epithelial cells after exposure to diesel exhaust micron sub particles ($PM_{1.0}$) and pollen allergens, Environ. Pollut. 171 (2012 Dec) 162–167.
[17] G. Mazzarella, V. Esposito, A. Bianco, F. Ferraraccio, M.V. Prati, A. Lucariello, L. Manente, A. Mezzogiorno, A. De Luca, Inflammatory effects on human lung epithelial cells after exposure to diesel exhaust micron sub particles ($PM_{1.0}$) and pollen allergens, Environ. Pollut. 161 (2012 Feb) 64–69.
[18] G. Mazzarella, A. Lucariello, A. Bianco, C. Calabrese, T. Thanassoulas, L. Savarese, A. Fiumarella, V. Esposito, A. De Luca, Exposure to submicron particles ($PM_{1.0}$) from diesel exhaust and pollen allergens of human lung epithelial cells induces morphological changes of mitochondria tonifilaments and rough endoplasmic reticulum, In Vivo 28 (4) (2014 07-08) 557–561.
[19] G. Mazzarella, F. Ferraraccio, M.V. Prati, S. Annunziata, A. Bianco, A. Mezzogiorno, G. Liguori, I.F. Angelillo, M. Cazzola, Effects of diesel exhaust particles on human lung epithelial cells: an in vitro study, Respir. Med. 101 (6) (2007 Jun) 1155–1162.
[20] G. Mazzarella, E. Grella, D. D'Auria, G. Paciocco, F. Perna, O. Petillo, G. Peluso, Phenotypic features of alveolar monocytes/macrophages and IL-8 gene activation by IL-1 and TNF-alpha in asthmatic patients, Allergy 55 (Suppl. 61) (2000) 36–41.
[21] G. Mazzarella, O. Petillo, S. Margarucci, C. Calabrese, G. Peluso, Role of monocyte/macrophage population in immune response, Monaldi Arch. Chest Dis. 53 (1) (1998 Feb) 92–96.
[22] E. Catena, G. Mazzarella, G.F. Peluso, P. Micheli, A. Cammarata, S.A. Marsico, Phenotypic features and secretory pattern of alveolar macrophages in atopic asthmatic patients, Monaldi Arch. Chest Dis. 48 (1) (1993) 6–15.
[23] M. Kang, N. Khandelwal, V. Ojili, et al., Multidetector CT angiography in pulmonary sequestration, J. Comput Assist. Tomogr. 30 (2006) 926–932.
[24] S. Fumino, N. Iwai, O. Kimura, S. Ono, K. Higuchi, Preoperative evaluation of the aberrant artery in intralobar pulmonary sequestration using multidetector computed tomography angiography, J. Pediatr. Surg. 42 (10) (2007) 1776–1779.
[25] A. Wang, T.A. D'Amico, M.F. Berry, Surgical management of congenital pulmonary malformations after the first decade of life, Ann. Thorac. Surg. 97 (6) (2014 Jun) 1933–1938.
[26] X. Huang, K. Sun, Y.D. Zhao, S.M. Vogel, Y. Song, N. Mahmud, Y.Y. Zhao, Human CD34+ progenitor cells freshly isolated from umbilical cord blood attenuate inflammatory lung injury following LPS challenge, PLoS One 9 (2) (2014 Feb 18) e88814.
[27] F. Moccia, S. Dragoni, F. Lodola, E. Bonetti, C. Bottino, G. Guerra, U. Laforenza, V. Rosti, F. Tanzi, Store-dependent Ca2+ entry in endothelial progenitor cells as a perspective tool to enhance cell-based therapy and adverse tumour vascularisation, Curr. Med. Chem. 19 (34) (2012 Dec 1) 5802–5818.
[28] Sanchez-Hernandez, U. Laforenza, E. Bonetti, J. Fontana, S. Dragoni, M. Russo, J.E. Avelino-Cruz, S. Schinelli, D. Testa, G. Guerra, V. Rosti, F. Tanzi, F. Moccia, Store operated Ca^{2+} entry is expressed in human endothelial progenitor cells, Stem Cells Dev. 19 (12) (2010 Dec) 1967–1981.
[29] F. Moccia, F. Lodola, S. Dragoni, E. Bonetti, C. Bottino, G. Guerra, U. Laforenza, V. Rosti, Tanzi F.Ca2+ signalling in endothelial progenitor cells: a novel means to improve cell-based therapy and impair tumour vascularisation, Curr. Vasc. Pharmacol. 12 (1) (2014 Jan) 87–105.
[30] R. Berra-Romani, J.E. Avelino-Cruz, A. Raqeeb, A. Della Corte, M. Cinelli, S. Montagnani, G. Guerra, F. Moccia, F. Tanzi, Ca2+-dependent nitric oxide release in the injured endothelium of excised rat aorta: a promising mechanism applying in vascular prosthetic devices in aging patients, BMC Surg. 13 (Suppl. 2) (2013 Oct 8) S40.
[31] F. Moccia, S. Dragoni, M. Cinelli, S. Montagnani, B. Amato, V. Rosti, G. Guerra, F. Tanzi, How to utilize Ca2+ signals to rejuvenate the repairative phenotype of senescent endothelial progenitor cells in elderly patients affected by cardiovascular diseases: a useful therapeutic support of surgical approach? BMC Surg. 13 (Suppl. 2) (2013 Oct 8) S46.
[32] C.F. Andrade, H.P. Ferreira, G.B. Fischer, Congenital lung malformations, J. Bras. Pneumol. 37 (2011) 259–271.
[33] D. Gonzalez, J. Garcia, E. Fieira, et al., Video-assisted thoracoscopic lobectomy in the treatment of intralobar pulmonary sequestration, Interact. Cardiovasc. Thorac. Surg. 12 (2011) 77–79.
[34] S. Hirai, Y. Hamanaka, N. Mitsui, et al., Surgical treatment of infected intralobar pulmonary sequestration: a collective review of patients older than 50 years reported in the literature, Ann. Thorac. Cardiovasc. Surg. 13 (2007) 331–334.
[35] L.M. Marine, F.E. Valdes, R.M. Mertens, et al., Endovascular treatment of symptomatic pulmonary sequestration, Ann. Vasc. Surg. 25 (2011) 696.e11–696.e15.
[36] R.A. Gustafson, G.F. Murray, H.E. Warden, R.C. Hill, G.E. Rozar, Intralobar sequestration a missed diagnosis, Ann. Thorac. Surg. 47 (6) (1989 Jun) 841–847.
[37] T. Okamoto, D. Masuya, T. Nakashima, et al., Successful treatment for lung cancer associated with pulmonary sequestration, Ann. Thorac. Surg. 80 (2005) 2344–2346.
[38] A. Bianco, S.K. Sethi, J.T. Allen, R.A. Knight, M.A. Spiteri, Th2 cytokines exert a dominant influence on epithelial cell expression of the major group human rhinovirus receptor, ICAM-1, Eur. Respir. J. 12 (3) (1998 Sep) 619–626.
[39] S.C. Whiteman, A. Bianco, R.A. Knight, M.A. Spiteri, Human rhinovirus selectively modulates membranous and soluble forms of its intercellular adhesion molecule-1 (ICAM-1) receptor to promote epithelial cell infectivity, J. Biol. Chem. 278 (14) (2003 Apr 4) 11954–11961.
[40] A. Bianco, S.C. Whiteman, S.K. Sethi, J.T. Allen, R.A. Knight, M.A. Spiteri, Expression of intercellular adhesion molecule-1 (ICAM-1) in nasal epithelial cells of atopic subjects: a mechanism for increased rhinovirus infection? Clin. Exp. Immunol. 121 (2) (2000 Aug) 339–345.
[41] S.K. Sethi, A. Bianco, J.T. Allen, R.A. Knight, M.A. Spiteri, Interferon-gamma (IFN-gamma) down-regulates the rhinovirus-induced expression of intercellular adhesion molecule-1 (ICAM-1) on human airway epithelial cells, Clin. Exp. Immunol. 110 (3) (1997 Dec) 362–369.
[42] E. Micillo, A. Bianco, D. D'Auria, G. Mazzarella, G.F. Abbate, Respiratory infections and asthma, Allergy 55 (Suppl. 61) (2000) 42–45.
[43] A. Vatrella, S. Montagnani, C. Calabrese, R. Parrella, G. Pelaia, G.L. Biscione, N. Corcione, S.A. Marsico, G. Guerra, Neuropeptide expression in the airways of COPD patients and smokers with normal lung function, J. Biol. Reg. Homeost. Agents 24 (4) (2010 Oct-Dec) 425–432.
[44] G. de Laurentiis, D. Paris, D. Melck, P. Montuschi, M. Maniscalco, A. Bianco, M. Sofia, A. Motta, Separating smoking-related diseases using NMR-based metabolomics of exhaled breath condensate, J. Proteome Res. 12 (3) (2013 Mar 1) 1502–1511.
[45] S. Dragoni, U. Laforenza, E. Bonetti, F. Lodola, C. Bottino, G. Guerra, A. Borghesi, M. Stronati, V. Rosti, F. Tanzi, F. Moccia, Canonical transient receptor potential 3 channel triggers VEGF-induced intracellular ca2+ oscillations in endothelial progenitor cells isolated from umbilical cord blood, Stem Cells Dev. 22 (19) (2013 Oct 1) 2561–2580.
[46] S. Dragoni, U. Laforenza, E. Bonetti, F. Lodola, C. Bottino, R. Berra-Romani, G. Carlo Bongio, M.P. Cinelli, G. Guerra, P. Pedrazzoli, V. Rosti, F. Tanzi, F. Moccia, Vascular endothelial growth factor stimulates endothelial colony forming cells proliferation and tubulogenesis by inducing oscillations in intracellular Ca2+ concentration, Stem Cells 29 (11) (2011 Nov) 1898–1907.
[47] F. Lodola, U. Laforenza, E. Bonetti, D. Lim, S. Dragoni, C. Bottino, H.L. Ong, G. Guerra, C. Ganini, M. Massa, M. Manzoni, I.S. Ambudkar, A.A. Genazzani, V. Rosti, P. Pedrazzoli, F. Tanzi, F. Moccia, C. Porta, Store operated Ca(2+) entry

is remodelled and controls in vivo angiogenesis in endothelial progenitor cells isolated from tumoural patients, PLoS One 7 (9) (2012) e42541.
[48] G. Shapovalov, R. Skryma, N. Prevarskaya, Calcium channels and prostate cancer, Recent Pat. Anticancer Drug Discov. 8 (1) (2013) 18–26.
[49] S. Dragoni, U. Laforenza, E. Bonetti, M. Reforgiato, V. Poletto, F. Lodola, C. Bottino, D. Guido, A. Rappa, S. Pareek, M. Tomasello, M.R. Guarrera, M.P. Cinelli, A. Aronica, G. Guerra, G. Barosi, F. Tanzi, V. Rosti, F. Moccia, Enhanced expression of Stim, Orai, and TRPC transcripts and proteins in endothelial progenitor cells isolated from patients with primary myelofibrosis, PLoS One 9 (3) (2014 Mar 6) e91099.